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Evaluation of fungicides against *Macrophomina phaseolina* caused by dry root rot of safflower

MD Navale, VM Gholve and GS Pawar

Abstract
Safflower (*Carthamous tinctorius* L.), is one of the most important *Rabi* oilseed crop of the family *compositae* or *asteraceae* in India. It is affected by several fungal diseases among them the dry root rot caused by *Macrophomina phaseolina* which cause severe economic yield loss. An attempt was made to manage the disease with fungicides. *In vitro* efficacy of seven systemic fungicides @ 500 and 1000 ppm, seven Non-systemic fungicides @1500 and 2000 ppm and seven combi-fungicides @ 1500 and 2000 ppm tested *in vitro* were found effective with significant mycelial growth inhibition of the test pathogen, over untreated control. However, resulted with cent percent inhibition Carbendazim 50% WP (100% and 100%), Mancozeb (86.67% and 94.00%), Carbendazim 25% + Mancozeb 50% (100% and 100%) and Carbendazim 12% + Mancozeb 63% (92.03% and 100%) were found most effectives against *M. phaseolina.*

Keywords: Safflower, Dry root rot, *Macrophomina phaseolina*, Fungicides

Introduction
Safflower (*Carthamous tinctorius* L.), is one of the most important *Rabi* oilseed crop of the family *compositae* or *asteraceae* in India. Safflower crop can be grown in wide range of soils like clay loam, sandy loam, shallow and light textured soils. This crop has being cultivated in tropical as well as in sub-tropical conditions with ideal temperature required for this crop being 22 ℃ to 35 ℃. It is popular among the farmers due to its hardy nature, short duration and high commercial value. Safflower crop suffers from fungal, bacterial and viral diseases. Dry root rot is a serious disease of safflower causing economic losses to growers in India and other countries. In India, dry root rot disease attack in most safflower growing areas and cause losses 42 to 45 per cent.

India ranks first in World in respect area and production of safflower. In India Maharashtra, Karnataka, Andhra Pradesh, Madhya Pradesh, Gujarat, Orissa and Bihar are major safflower growing states. In 2018-19 and 2019-20 India area 46.00 and 52.00 ('000 ha), production 25.00 and 44.00 ('000 Tonne) and productivity 537 and 843 kg/ ha. Maharashtra ranks first in India in respect area and production of safflower. In 2019-20 Maharashtra state area is 21.60 ('000 ha), production 14.93 ('000 Tonne) and productivity 691 kg/ha. (Anonymous 2020a & 2020b) [2, 3].

The diseases of safflower, among these diseases, root rot caused by *Macrophomina phaseolina* (Tassi) Goid is a very devastating disease of safflower and causes heavy reduction in total yield (Kore and Deshmukh, 1982) [10]. It is the major soil borne disease and appears sporadically all over the country (Shambharkar and Indi, 1987) [17]. Occurrence of this disease on safflower in India was first reported by Amarsingh and Bhowmik (1979) [1] from IARI, New Delhi and later on by-others from different parts of the country (Kore and Deshmukh, (1982) [10] and Lukade, (1992) [11] from Maharashtra; Singh *et al.*, (1987) [18] from M.P).

Materials and Methods:
Collection and isolation of the pathogenic isolates:
The field was survey carried out during 2018-19 and 2019-20, a large number of infected safflower roots were collected from 8 different districts from Marathwada region. Place *viz*., Aurangabad, Beed, Hingoli, Jalna, Latur, Nanded, osmanabad and Parbhani districts. These samples were subjected to standard tissue isolation. The safflower roots showing typical bark feeling and disintegrated roots were cut into small bits measuring about 2 mm and surface sterilized in ($HgCl_2$) (0.1%) for one minute such bits were transferred to Petri dishes

containing sterile water successively for three times and then into the Petri dishes containing sterile water successively for three times and then into the Petri dishes containing 20 ml of potato dextrose agar (PDA) medium and incubated at ± 28 ℃ for 10 days and observed for fungal growth. The culture of *M. phaseolina* was maintained at 5 ℃ in the refrigerator and sub cultured periodically at an interval of 20 to 25 days during the course of the investigation.

Efficacy of different fungicides against *Macrophomina phaseolina*: *In vitro* efficacy of seven fungicides were evaluated systemic @ 500 and 1000 ppm, Non-systemic fungicides were (@ 1500 and 2000 ppm) and Combi fungicides were (@ 1500 and 2000 ppm) conc against *M. phaseolina* (MpH_3 isolate), by Poisoned food technique (Nene and Thapliyal, 1993) [15]. The pathogen *M. phaseolina* was grown on PDA medium in Petri-plates for ten days prior to setting up the experiment. Fungicide suspension was prepared in PDA by adding required quantity of fungicide to obtain the desired concentration on the basis of active ingredient present in the chemical. Twenty ml of poisoned medium was poured in each of the sterilized Petri plates. Mycelial disc of 5 mm was taken from the periphery of nine day old culture and placed in the centre and incubated at 28±2 ℃ till growth of the fungus touched the periphery in control plate. Suitable checks were also maintained without addition of any fungicide, three replications were maintained for each treatment. The diameter of the colony was measured in two directions and average was worked out. The percent mycelial inhibition was calculated by using the formula given by Vincent (1927) [20] and data were analyzed statistically by using Completely Randomized Design (CRD).

$$\text{Per cent inhibition} = \frac{C-T}{C} \times 100$$

Where,
C= growth of the test pathogen in untreated control plates (mm)
T= growth of the test pathogen a in treated plates (mm)

Result and Discussion

In vitro efficacy of systemic fungicides

Mycelial growth

Results (Table 1, Plate 1 and Fig. 1) revealed that all of the seven systemic fungicides tested exhibited a wide range of radial mycelial growth of *M. phaseolina* and it was decreased drastically with increase in their concentrations.

At 500 ppm, radial mycelial growth was ranged from 00.00 mm (Carbendazim) to 32.04 mm (Thiophanate methyl). However, with Carbendazim 50% WP there was complete inhibition of the mycelial growth which was significantly superior over all the treatments. The next fungicide with significantly least mycelial growth was Difenconazole 25% EC (8.49 mm) followed by Propiconazole 25% EC (14.92 mm), Hexanconazole 5% EC (18.67 mm), Tebuconazole 29.9% EC (21.85 mm), Benomyl 50% WP (21.96 mm) Thiophanate methyl 70% WP (32.04 mm).

At 1000 ppm, similar trend as that of 500 ppm was observed and radial mycelial growth ranged from 00.00 mm (Carbendazim) to 28.19 mm (Thiophanate methyl). However, with Carbendazim 50% WP there was complete inhibition of the mycelial growth and significantly superior over all the treatments. The next fungicides with significantly least mycelial growth were Difenconazole 25% EC (5.06 mm) followed by Propiconazole 25% EC (12.26 mm), Benomyl 50% WP (13.43 mm), Hexanconazole 5% EC (15.54 mm), Tebuconazole 29.9% EC (15.80 mm) and Thiophanate methyl 70% WP (28.13 mm).

Table 1: *In vitro* efficacy of systemic fungicides against mycelial Growth and inhibition of *M. phaseolina* (MpH_3)

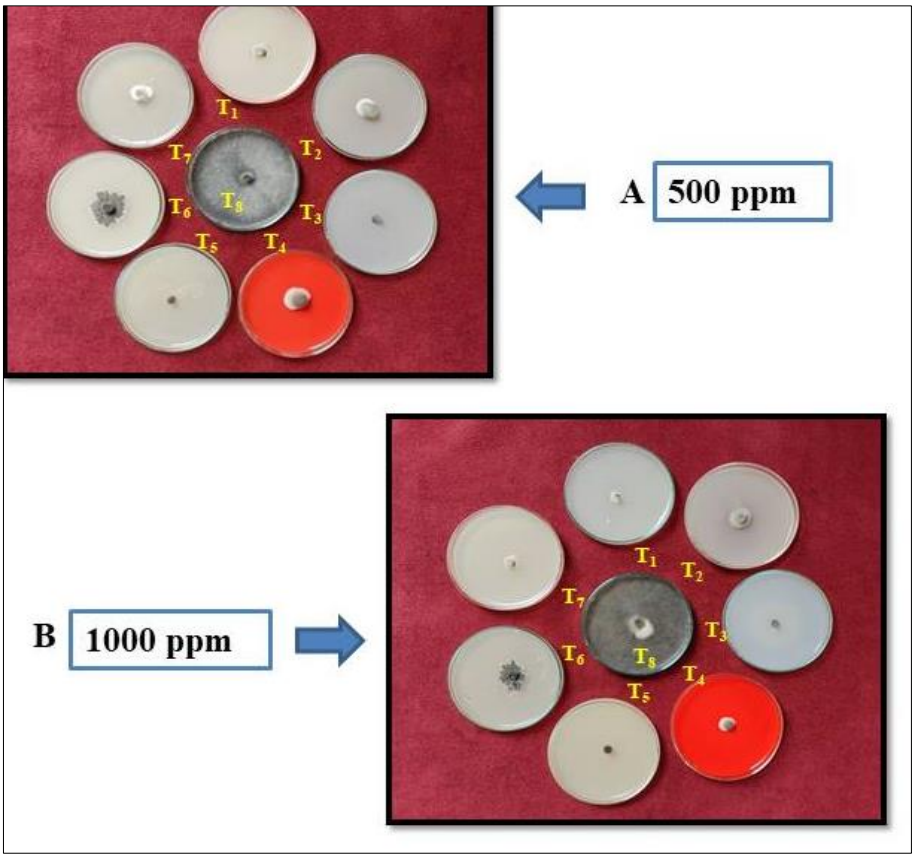
Tr. No.	Treatments	Mean Colony Dia. *(mm) at conc.		% Inhibition* at ppm	
		500 ppm	1000 ppm	500 ppm	1000 ppm
T_1	Propiconazole 25% EC	14.92	12.26	83.42 (65.97)	86.38 (68.34)
T_2	Hexaconazole 5% EC	18.67	15.54	79.26 (62.91)	82.73 (65.45)
T_3	Difenconazole 25% EC	8.49	5.06	90.57 (72.11)	94.38 (76.28)
T_4	Tebuconazole 29.9% EC	21.85	15.80	75.72 (60.48)	82.44 (65.23)
T_5	Carbendazim 50% WP	0.00	0.00	100.00 (90.00)	100.00 (90.00)
T_6	Thiophanate methyl 70% WP	32.04	28.13	64.40 (53.37)	68.74 (56.01)
T_7	Benomyl 50% WP	21.96	13.43	75.60 (60.40)	85.08 (67.28)
T_8	Control (untreated)	90.00	90.00	0.00 (0.00)	0.00 (0.00)
SE (m) ±		0.31	0.25	0.17	0.15
C.D. (P=0.01)		0.93	0.75	0.51	0.46

*: Mean of three replications, Dia: Diameter Figures in parentheses are arcsine transformed values

Mycelial inhibition

Results (Table 1, Plate 1 and Fig. 1) revealed that all the systemic fungicides tested (each @ 500 and 1000 ppm) significantly inhibited mycelial growth of *M. phaseolina*, over untreated control. Further, percent mycelial inhibition was increased with increase in concentrations of the fungicides tested (Fig. 1).

At 500 ppm, mycelial growth inhibition was ranged from 64.40 (Thiophanate methyl) to 100 (Carbendazim) per cent. However, Carbendazim 50% WP gave cent percent (100%) mycelial inhibition which was significantly superior over all the treatments followed by Difenconazole 25% EC (90.57%), Propiconazole 25% EC (83.42%), Hexanconazole 5% EC (79.26%), Tebuconazole 29.9% EC (75.72%) and Benomyl 50% WP (75.60%).



Tr. No.	Treatments	Tr. No.	Treatments
T1	Propiconazole 25% EC	T5	Carbendazim 50% WP
T2	Hexaconazole 5% EC	T6	Thiophanate methyl 70% VIP
T3	Difenconazole 25% EC	T7	Benomyl 50% NP
T4	Tebuconazole 29.9% EC	T8	Control (untreated)

Plate 1: A, B. *In vitro* efficacy of systemic fungicides @ 500 and 1000 ppm *against M. phaseolina* (MpH3 isolate)

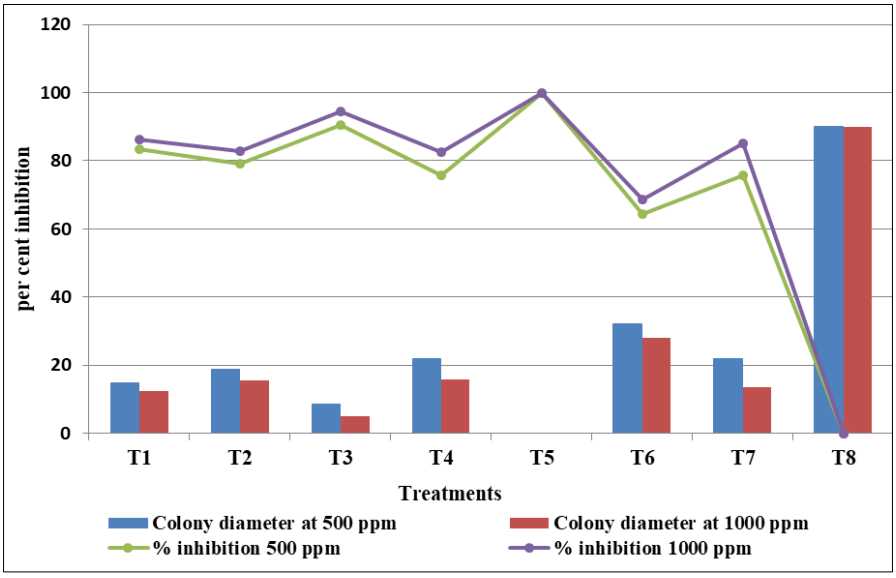


Fig 1: *In vitro* bioefficacy of systemic fungicides against *M. phaseolina*

At 1000 ppm, same trend as at 500 ppm was observed and mycelial growth inhibition ranged from 68.74 (Thiophanate methyl) to 100 (Carbendazim) per cent. However, Carbendazim gave cent percent (100%) mycelial inhibition followed by Difenconazole 25% EC (94.38%), Propiconazole 25% EC (86.38%), Benomyl 50% WP (85.08%), Hexanconazole 5% EC (82.73%) and Tebuconazole 29.9% EC (82.44%).

***In vitro* evaluation of non-systemic / contact fungicides**

Radial mycelial growth

Results (Table 2, Plate 2 and Fig. 2) revealed that all the Non-

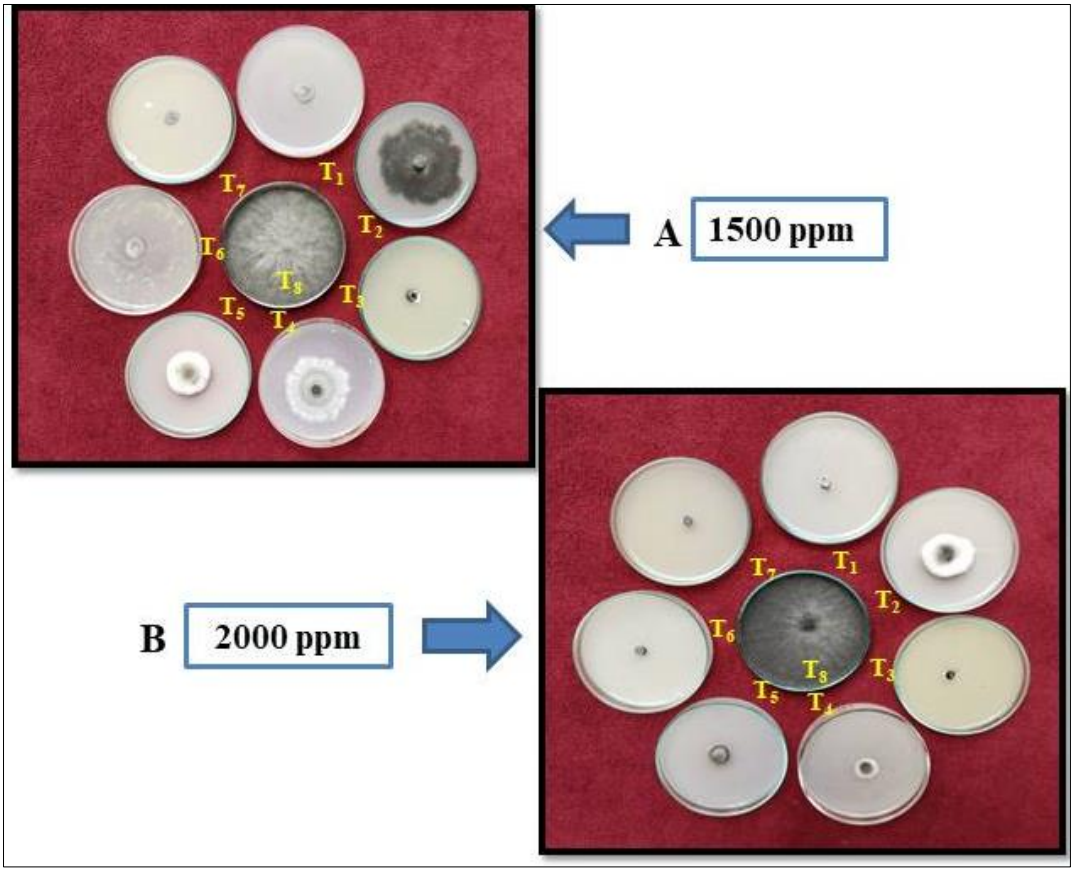
systemic fungicides tested (each @ 1500 and 2000 ppm) significantly inhibited mycelial growth of *M. phaseolina*, over untreated control. Further, percent mycelial inhibition was increased with increase in concentrations of the fungicides tested (Fig. 4.15).

At 1500 ppm, radial mycelial growth was ranged from 12.00 mm (Mancozeb) to 65.03 mm (Copper oxychloride). However, it was significantly least with Mancozeb (12.00 mm) which was significantly superior over all the treatments followed by Thiram (17.07 mm), Propineb (19.86 mm), Copper hydroxide (25.90 mm), Chlorothalonil (35.00 mm), Captan (54.00 mm) and Copper oxychloride (65.03 mm).

Table 2: *In vitro* efficacy of non-systemic fungicides against mycelial growth and inhibition of *M. phaseolina* (MpH_3)

Tr. No.	Treatments	Colony Dia.* (mm) at conc.		% Inhibition* at ppm	
		1500 ppm	2000 ppm	1500 ppm	2000 ppm
T_1	Copper hydroxide	25.90	14.12	71.22 (57.56)	84.31 (66.67)
T_2	Copper oxychloride	65.03	35.46	27.74 (31.78)	60.60 (51.12)
T_3	Mancozeb	12.00	5.40	86.67 (68.58)	94.00 (75.82)
T_4	Captan	54.00	17.00	40.00 (39.23)	81.11 (64.24)
T_5	Chlorothalonil	35.00	16.46	61.11 (51.42)	81.71 (64.68)
T_6	Propineb	19.86	13.38	77.93 (61.98)	85.13 (67.32)
T_7	Thiram	17.07	10.94	81.03 (64.18)	87.84 (69.60)
T_8	Control (untreated)	90.00	90.00	0.00	0.00
SE (m) ±		0.30	0.25	0.11	0.14
C.D. (P=0.01)		0.90	0.77	0.34	0.42

*Mean of three replications. Dia: Diameter; Figures in parenthesis are arc sine transformed value



Tr. No.	Treatments	Tr. No.	Treatments
T:1	Carboxin	T:5	agorothalonil
T:2	Copper oxychloride	T:6	Propineb
T:3	Mancozeb	T:7	Milt
T:4	Captan	T:8	Control (untreated)

Plate 2: A, B. *In vitro efficacy* of Non-systemic fungicides *Cr* 1500 and 2000 ppm against .11. *Phaseolina* (MpH3 isolate)

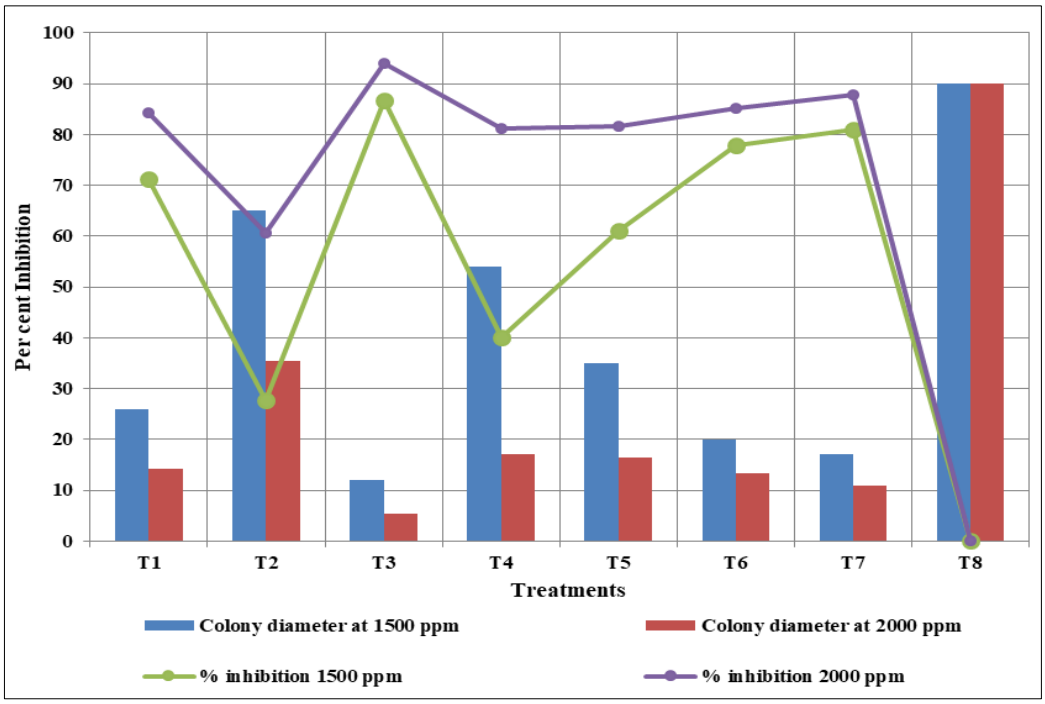


Fig 2: *In vitro* bioefficacy of Non-systemic fungicides against *M. phaseolina*

At 2000 ppm, similar trend as that of 1500 ppm was observed and radial mycelial growth ranged from 5.40 mm (Mancozeb) to 35.46 mm (Copper oxychloride). However, it was significantly least with Mancozeb (5.40 mm) followed by Thiram (10.94 mm), Propineb (13.38 mm), Copper hydroxide (14.12 mm), Chlorothalonil (16.46 mm), Captan (17.00 mm) and Copper oxychloride (35.46 mm).

Mycelial inhibition

Results (Table 2, Plate 2 and Fig. 2) revealed that all the Non-systemic fungicides tested (each @ 1500 and 2000 ppm) significantly inhibited mycelial growth of *M. phaseolina*, over untreated control. Further, percent mycelial inhibition was increased with increase in concentrations of the fungicides tested (Fig. 4.15).

At 1500 ppm, mycelial growth inhibition was ranged from 27.74 (Copper oxychloride) to 86.67 (Mancozeb) per cent. However, Mancozeb gave 86.67 percent mycelial inhibition which was significantly superior over all the treatments followed by Thiram (81.09%), Propineb (77.93%), Copper hydroxide (71.22%), Chlorothalonil (61.11%), Captan (40.00%) and Copper oxychloride (27.74%).

At 2000 ppm, mycelial growth inhibition was ranged from 60.60 (Copper oxychloride) to 94.00 (Mancozeb) per cent. However, Mancozeb gave 94.00 percent mycelial inhibition followed by Thiram (87.84%), Propineb (85.13%), Copper hydroxide (84.31%), Chlorothalonil (81.71%), Captan (81.11%) and Copper oxychloride (60.60%).

***In vitro* evaluation of combi- fungicides**

Radial mycelial growth

Results (Table 3, Plate 3 and Fig. 3) revealed that all of the seven combi-fungicides tested exhibited a wide range of radial mycelial growth of *M. phaseolina* and was decreased drastically with increase in concentrations of the test fungicides from 1500 to 2000 ppm.

At 1500 ppm, radial mycelial growth of the test pathogen ranged from 0.00 mm (Carbendazim 25% + Mancozeb 50% WS) to 69.00 mm (Metalaxyl 8% + Mancozeb 64% WP). However, it was significantly least with Carbendazim 12% + Mancozeb 63% WP (7.17 mm) and found significantly superior over all the treatments followed by Carboxine 37.5% + Thiram 37.5% (9.55 mm), Metalaxyl M 4% + Mancozeb 64% (13.83 mm), Hexaconazole 4% + Zineb 68% (20.00 mm), Trifloxystrobin 25% + Tebuconazole 50% (25.92 mm) and Metalaxyl 8% + Mancozeb 64% (69.00 mm).

Table 3: *In vitro* efficacy of Combi fungicides against mycelial growth and inhibition of *M. phaseolina* (MpH_3)

Tr. No.	Treatments	Mean Colony Dia. *(mm) at conc.		% Inhibition* at ppm	
		1500 ppm	2000 ppm	1500 ppm	2000 ppm
T_1	Carbendazim 12% + Mancozeb 63%	7.17	0.00	92.03 (73.61)	100.00 (90.00)
T_2	Hexaconazole 4% + Zineb 68%	20.00	16.43	77.78 (61.87)	81.74 (64.71)
T_3	Trifloxystrobin 25% + Tebuconazole 50%	25.92	20.44	71.20 (57.54)	77.29 (61.54)
T_4	Carboxine 37.5% + Thiram 37.5%	9.55	8.83	89.39 (70.99)	90.19 (71.75)
T_5	Carbendazim 25% + Mancozeb 50%	0.00	0.00	100.00 (90.00)	100.00 (90.00)
T_6	Metalaxyl 8% + Mancozeb 64%	69.00	60.90	23.33 (28.88)	32.33 (34.65)
T_7	Metalaxyl M 4% + Mancozeb 64%	13.83	10.50	84.63 (66.92)	88.33 (70.03)
T_8	Control (untreated)	90.00	90.00	0.00 (0.00)	0.00 (0.00)
SE (m) ±		0.27	0.17	0.21	0.17
C.D. (P=0.01)		0.81	0.50	0.63	0.51

*Mean of three replications. Dia: Diameter; Figures in parenthesis are arc sine transformed value

At 2000 ppm, radial mycelial growth of the test pathogen ranged from 0.00 mm (Carbendazim 25% + Mancozeb 50% WS and Carbendazim 12% + Mancozeb 63%) to 60.90 mm (Metalaxyl 8% + Mancozeb 64% WP). However, it was significantly least with Carboxine 37.5% + Thiram 37.5% (8.83 mm), Metalaxyl M 4% + Mancozeb 64% (10.50 mm), Hexaconazole 4% + Zineb 68% (16.43 mm), Trifloxystrobin 25% + Tebuconazole 50% (20.44 mm) and Metalaxyl 8% + Mancozeb 64% (60.90 mm).

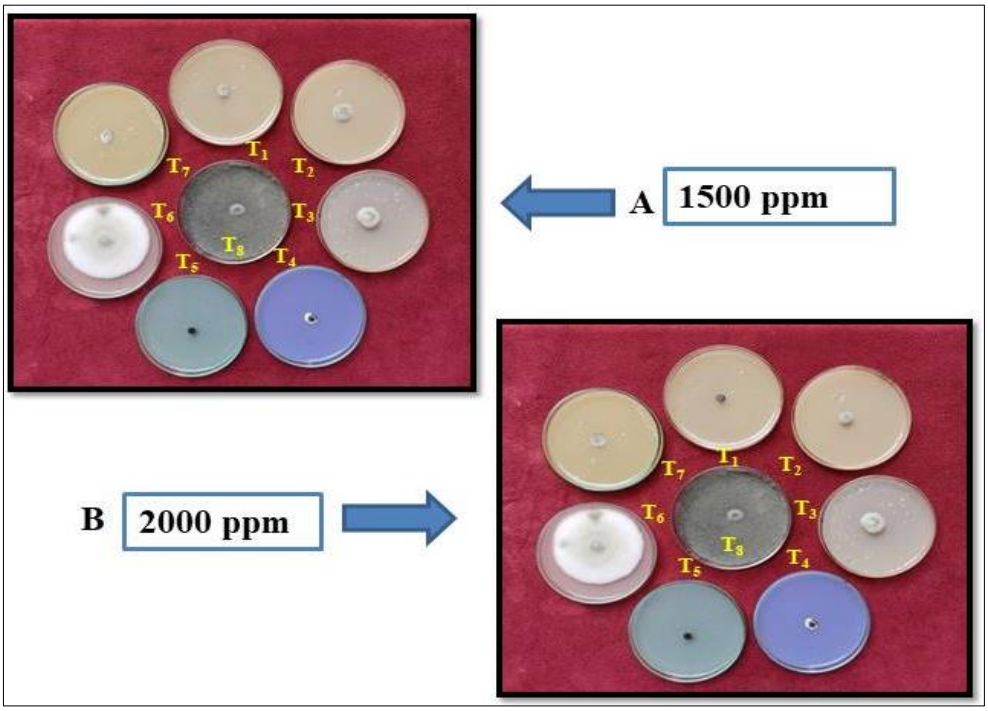
Mycelial inhibition

Results (Table 3, Plate 3 and Fig. 3) revealed that all the combi fungicides tested (each @ 1500 and 2000 ppm) significantly inhibited mycelial growth of *M. phaseolina*, over untreated control. Further, percent mycelial inhibition was increased with increase in concentrations of the fungicides tested (Fig. 4.18).

At 1500 ppm, mycelial growth inhibition was ranged from 23.33 percent (Metalaxyl 8% + Mancozeb 64%) to 100.00 percent (Carbendazim 25% + Mancozeb 50% WS. However, Carbendazim 25% + Mancozeb 50% WS and Carbendazim 12% + Mancozeb 63% completely 100.00 percent mycelial inhibition which was significantly superior over all the treatments followed Carboxine 37.5% + Thiram 37.5% (89.39%), Metalaxyl M 4% + Mancozeb 64% (84.63%), Hexaconazole 4% + Zineb 68% (77.78%), Trifloxystrobin 25% + Tebuconazole 50% (71.20%) and Metalaxyl 8% + Mancozeb 64% (23.33%).

At 2000 ppm, mycelial growth inhibition was ranged from 32.33 (Metalaxyl 8% + Mancozeb 64%) to 100.00 (Carbendazim 25% + Mancozeb 50% WS and Carbendazim 12% + Mancozeb 63%) per cent. However, Carbendazim 25% + Mancozeb 50% WS and Carbendazim 12% + Mancozeb 63% completely 100.00 percent mycelial inhibition followed by Carboxine 37.5% + Thiram 37.5% (90.19%), Metalaxyl M 4% + Mancozeb 64% (88.33%), Hexaconazole 4% + Zineb 68% (81.74%), Trifloxystrobin 25% + Tebuconazole 50% (77.29%) and Metalaxyl 8% + Mancozeb 64% (32.33%).

Thus, all the fungicides tested were found fungistatic against *M. phaseolina* significantly inhibited its mycelial growth over untreated control. However, fungicides found most effective were systemic fungicides *viz.*, Carbendazim, Difenconazole, Propiconazole, and Hexaconazole. Non-systemic fungicides *viz.*, Mancozeb, Thiram, Propineb, Copper hydroxide. Combi fungicides *viz.*, Carbendazim 25% + Mancozeb 50%, Carbendazim 12% + Mancozeb 63% , Carboxine 37.5% + Thiram 37.5% , Metalaxyl M 4% + Mancozeb 64%.



Tr. no.	Treatments	Tr. No.	Treatments
T1	Carbendazim 12% + Mancozeb 63%	T5	Carbendazim 25 % +Mancomb 50 %
T2	Hexaconazole 4% + tab 68%	T6	Metalaxyl 8 % + Mancozeb 64%
T3	Ttifloxystrobin 25% + Tebneonazole 50%	T7	Metalaxyl M 4% + Mancozeb 64%
T4	Cabo.* 37.5% + Tbiram 37.5%	T8	Control (untreated)

Plate 3: A, B. *In vitro* efficacy of Combi ftutocides @ 1500 and 2000 ppm against *M. phaseolina* (MpH_3 isolate)

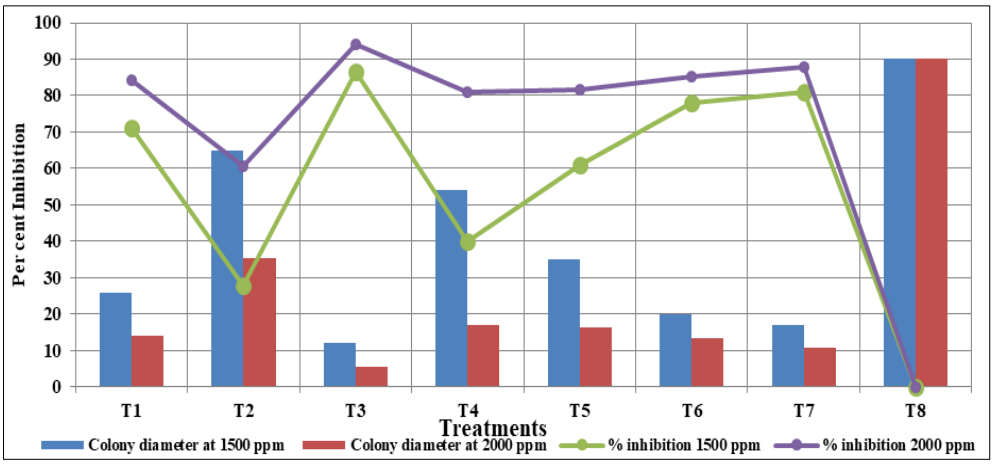


Fig. 3: *In vitro* bioefficacy of Combi fungicides against *M. phaseolina*

These findings are in conformity with the earlier findings of those workers who reported these fungicides had significantly inhibited mycelial growth of *M. phaseolina*/*R. bataticola* causing dry root rot/charcoal rot of safflower as well as many other crops. Khonde *et al.*, (2008) [9] as Carbendazim + Thiram (0.1 + 0.2%), Penconazole (0.1%) and Thiophanate-M (0.1%) most effective in inhibiting mycelia growth of *R. bataticola*, causing root rot of soybean; Magar *et al.*, (2011) [12] reported the fungicides *viz*., Carbendazim 50% WP @ 0.1% and Mancozeb 75% WP @ 0.25% caused 100 percent mycelia growth inhibition of *M. Phaseolina*; the fungicides Difenconazole 25% EC and Benomyl 50% WP (each @ 0.1%) were also reported effective; Moradia, (2011) [15] reported that the fungicides *viz.*, Difencozole (Score 25% EC), Carboxine (Vitavax 75% WP) and carbendazium + mancozeb (SAFE 75 WP) were most effective which caused percent mycelial growth inhibition at all the concentrations; (Malathi and Doraisamy 2003 [13]; Khan *et al.*, 2012 [8]; Sangeetha and Jahagirdar 2013b [16]; Deshmukh *et al.* 2014 [6] Chaudhary *et al.*, 2017 [5]; Maruti *et al.*, 2017b [14]; Arvind and Brahmbhatt 2018 [4]; Sharma and Kumari 2018; Thombre and Kohire *et al.*, 2018b [19]; Kishanawat *et al.*, 2021[7]

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